Conclusion: Results encourage for further research of factors that balance injurious vs. protective and regenerative responses towards initial liver cell stress in order to develop new solutions for prevention and treatment of antituberculosis drug-induced hepatotoxicity complicated with concomitant diseases.

PP-044 Hydrodynamic-based delivery of plasmid DNA encoding c-met ameliorates D-galactosamine/lipopolysaccharide induced acute liver failure in rats

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Objectives: The absence of HGF receptor (c-met) in the rats with D-galactosamine/lipopolysaccharide treatment leads to the fact that high expression of HGF cannot activate signal transduction from c-met on survivor hepatocytes. Whether exogenous administration of c-met by gene transfer can promote liver regeneration and protects the rats from liver failure is not clear.
Methods: We established the rat model with acute liver failure induced by D-GalN/LPS, and constructed plasmid DNA encoding c-met with albumin promoter (p-alb-c-met), and then transfer it into the rat liver by hydrodynamic injection. The expression of c-met and PCNA in the liver was detected by immunohistochemical staining, and then apoptosis in hepatocytes was measured by flow cytometry.
Results: Hydrodynamic delivery of p-alb-c-met induced high and targeted expression of c-met in rat liver cells, which markedly reduced hepatocytes apoptosis, and promoted the proliferation of survivor hepatocytes, and then elevated significantly the survival rate among the rats with liver failure.

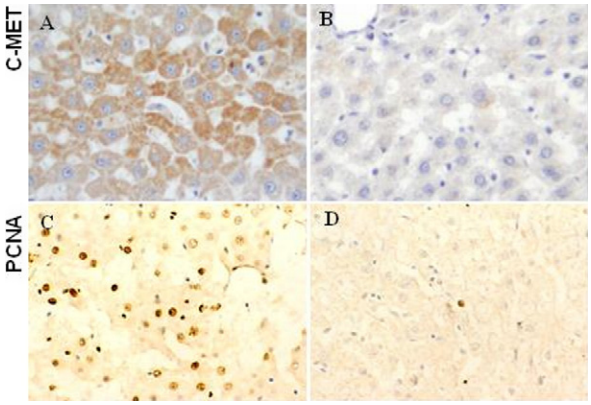

Figure 1. c-met expression was shown in pALB-C-MET-treated (A) and pALB-GFP-treated (B) rats 6 hours following administration of D-GalN/LPS. Rats expressing c-met (C) have a significant proliferative advantage over pALB-GFP-treated rats (D).

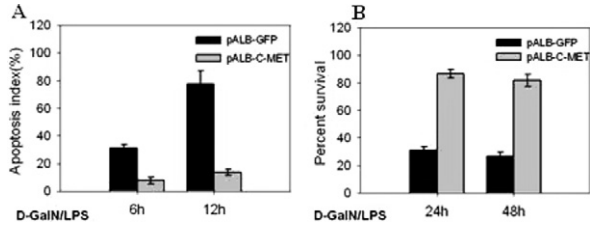

Figure 2. c-met markedly reduced hepatocytes apoptosis (A) and elevated the survival rate (B) among rats with D-GalN/LPS treatment.

Conclusions: These results qualify c-met as part of a physiologic, protective response of hepatocytes to injury and a promising gene therapy candidate for clinical applications aimed at preventing and treating acute liver failure.

PP-045 Exercise aggravated immune disorders and inflammation of rats in sulfur dioxide environment

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Objectives: Evidence suggests that increased air pollutants impacts with increased exercise activity levels. The present study aimed to determine the changes of IgA, IgG, IgM, IgE, TNF-α and IL-4 with exercise training and sulfur dioxide (SO_2) exposure in SD rats' serum, and investigate mechanisms of exercise decrease immune function in SO_2 environment.
Methods: Forty male SD rats were randomly divided into control group (CG, n = 10), exercise group (EG, n = 10), pollution plus rest group (PRG, n = 10) and pollution plus exercise group (PEG, n = 10). The rats were housed in exposure chambers (1m^3) and treated with different concentration of SO_2 (0mg/m^3, 10mg/m^3). EG and PEG performed exercise training with a motor-driven wheel (speed for 8 m/min 2 h/d for 10 d). The levels of IgA, IgG, IgM, IgE, TNF-α and IL-4 were estimated by radioimmunoassay. Differences in parameters among groups were compared by using one-way ANOVA test.
Results: The IgA and IgM levels were significantly decreased in PEG compare with PRG ($P < 0.05$); whereas the levels of IgE were significantly increased in PEG compare with PRG ($P < 0.01$); in additional, IL-4 and TNF-α levels were higher in PEG than PRG ($P < 0.05$). The mean±SD level of IgA in PRE was 0.335±0.068 mg/ml and PEG was 0.263±0.054 mg/ml. The mean±SD level of IgM in PRE was 0.448±0.154 mg/ml and PEG was 0.316±0.068 mg/ml. The mean±SD level of IL-4 in PRE was 10.413±2.325 ng/ml and PEG was 16.413±4.803 ng/ml. The mean±SD level of TNF-α in PRE was 21.411±3.676 ng/ml and PEG was 33.720±9.137 ng/ml.
Conclusion: These results show that exercise training aggravated immunological suppression and paroxysm of anaphylactic disease in rats on SO_2 exposure, and the serum changes of immunoglobulin and cytokines levels may play an important role in the pathogenesis.

PP-046 The repair effects of Pim-3 on liver in fulminant hepatic failure rat model

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Objective: To investigate the liver repair effects of Pim-3 gene in rats with fulminant hepatic failure (FHF).
Methods: Thirty-two rats were randomly divided into four groups (eight for each group). Three groups of rats were pretreated with Ringer's solution, vector plasmid and Pim-3 gene recombinant plasmid respectively and, one day later, received intraperitoneal injections with lipopolysacchride (LPS) and D-galactosamine (D-GalN). The fourth group served as normal control. Eight hours after the LPS/D-GalN injection, the liver tissues and blood samples were collected and tested by biochemistry, molecular biology, immunology and pathology methods. Comparisons between groups were done by analysis of variance.
Results: The over-expressions of Pim-3 gene and reporter gene, green fluorescent protein (GFP) were induced by